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THE GREEKS HAD A WORD FOR IT*

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AMONG the lesser deities worshiped by the ancient Greeks there was one who wore the beard, the ears and the horns of a goat. In fact, his imaginary aspect largely shaped our subsequent conception of the Devil. According to the Greeks, when this creature appeared suddenly to human beings, he awoke in them a storm of sudden fear. They called him Pan, and called the fear that he evoked "panic."

In this respect, in this trait of provoking panic or fear, the great god Pan has his prototype today; but we no longer worship the lesser gods, so we do not call him Pan. We call him "Doctor," and tremble at his name.

The prospect of summoning a doctor, for himself or for one of his family, frightens almost every layman. This may be — and doubtless is — absurd; but the fear is deeply rooted in the past — and the physician himself is its father. The old witch doctors who catered to savage tribes used masks, incantations and every imaginable sort of humbug to frighten their prospective patients. To observe their antics was to conclude that they believed a patient thoroughly frightened was already half cured. It is too often true today that doctors appear to proceed on the same theory. Their actions suggest that they believe a good fright beats all the drugs in the pharmacopoeia.

Doctors do not sufficiently realize the fact that their patients are frightened, and do not give enough attention to the particular branch of pathology that involves a study of the emotions. I know that you have courses in what is called "psychosomatic medicine," — the phrase alone, like most medical phrases, is enough to frighten the average patient, — yet I suspect that it is a doctor, not a patient, who gives those courses; and this may reduce their usefulness. During a recent brief wait in a doctor's office I took off his shelves a book dealing with some general principles of private practice, and found in it a chapter on psychiatry. That chapter dealt with mental aberrations resulting from drunkenness and from syphilis but with no other mental twists at all. Yet I suggest to you that every patient, sick or well, is in greater or less degree a psychoneurotic — and should be so considered by his doctor.

If this is true in no other respect, you may be sure he has a fear psychosis; for he is afraid of you. I suggest to you that in this respect your education has been neglected; that you have been insufficiently warned of the importance, when you begin to practice, of healing first of all your patient's fear.

Of course I understand — and so do you — that your medical education is just beginning. If you are wise, you will accept the fact that it will never be complete. You have learned something about the use of medicines, have witnessed or have performed dissections of dead and of living flesh and have had some contact with the sick. But your first private patient will extend your education, and — if your mental capacities endure that long — so will your last. In fact, I suspect that every medical school should have a course of lectures given by patients! Certainly any doctor, after having once been ill himself, is a better doctor afterward.

The lesson that every doctor most needs to learn is that his patients are afraid. They have heard so many dreadful tales beginning with the formula: "He never had a sick day in his life till one Friday he felt a little queer and went to a doctor, and do you know. . . ." If they are of my generation they have heard their elders say ruefully, "Yes, he's going to have an operation, and you know the knife is the knife." It is, basically, not the knife the layman fears; it is that he will die, as the phrase goes, "on the table." I believe that deaths during an operation are decidedly rare. If every prospective operative patient — and his family — knew this, if he were assured that, no matter what the long-range results might be, he would survive the actual operation, his fears would be tremendously relieved.

There are so many of these fears. The patient is afraid of what the doctor will tell him, and as a result he often puts off seeing a doctor at all till it is too late to help him. Basically this is not fear of what the doctor may discover, but fear of the mysterious and terrible and godlike creature himself. Your patients will be afraid of you. Of course if you

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are young and handsome and your patients are neurotic women, this fear may be replaced by another emotion; but the normal person, finding it necessary to see a doctor, is frightened. I believe that some study has been made of the body's chemical reaction to emotions. Those of you who incline to a life of research might go far in that field. If, for instance, ulcers of the stomach are caused by an excess of hydrochloric acid in the digestive fluid, what emotions produce that excess? If you always remember that a patient who comes to you for diagnosis is afraid, what allowance must you make, in appraising his symptoms, for the chemical effect on him of that fear? What steps can you take to allay it, to allay not only his fears, but those of the well people who love him?

For of course the wise doctor will always remember that it may be quite as easy to make a well person sick as to make a sick person well. The recital of symptoms may produce them — in the well or in the sick. To suggest a parallel in which the conscious replaces the subconscious: suppose a man goes to a lawyer, says, "I want to sue John Doe" and recites his grievance. The lawyer says to him, "You have no case." He asks, "Why not?" And the lawyer replies, "Well, to have a good case, John Doe must have done and said so and so, and you must have done and said so and so." The client, thus informed of what his evidence should be, will go into court and testify to the exact state of affairs thus suggested to him. He will soon come to believe that he is telling the truth. In the same way the patient to whom symptoms are suggested will as often as not imagine he has them.

Probably a large proportion of you are planning to become specialists — and you have doubtless heard of the young doctor who appealed to a wise old physician for advice as to which specialty he should adopt. The old man suggested skin diseases, the young man asked why, and the old man said, "Because anyone who consults a doctor about a skin disease can afford to pay his bill; and patients with skin diseases don't die of them; and they don't get better." I always thought this anecdote amusing till I acquired a little purpura of my own.

Some of you will become specialists. For those of you who do so, the fact that your patient is afraid of you is particularly worth remembering; for he is much more afraid of you than of his family physician. For one thing, you and he are usually strangers; for another, he mistrusts you because he knows that a specialist, like a shoemaker, tends to stick to his last, to his specialty.

I heard an instance of this a week ago. At a certain medical school, specialists came regularly to lecture; and for material to illustrate the points they wished to make they turned to the wards in the available hospital. Once upon a time a psychiatrist came to lecture, found in the wards a patient with a pronounced psychosis and used her as a text for his discourse. A fortnight later a dermatologist found in the wards a sufferer from numerous skin lesions, and used this patient to illustrate his points. Subsequently an expert on digestive disturbances selected a patient with persistent diarrhea, and since she had spent twenty years in China, the students made elaborate studies of her stools to discover the cause. No one was struck by the coincidence that the patient who was used by each man as an object lesson was the same person until a general practitioner, an old country doctor, happened on her and after a glance at her history said in some excitement: "Why here you have something really remarkable. Dementia, dermatitis, diarrhea! This woman has pellagra." Which was true.

This tendency to wear blinders, to see only his own field, is one of the specialist's handicaps. His achievements are the more remarkable because he struggles against so many of these handicaps — handicaps rooted in his own psychology, handicaps arising from his inevitable ignorance of his patients, handicaps arising out of the fact that he knows his own field — or that he doesn't!

The increasing tendency toward specialization has led medicine to follow the example of industry,— to establish a production line. The human chassis, on a belt conveyor, passes under the eyes of a regiment of specialists, and one does this and another does that, each restricting himself to his particular field. And as though the mechanic who affixed the bumper thought he had built the car, each doctor when the patient recovers may secretly assure himself that he and he alone accomplished the cure. For the specialist tends to find in his own specialty the source of every human ill. To the dentist, it is the teeth; to the oculist, it is the eye; to the gastroenterologist, the stomach; to the cardiologist, the heart. Just as the successful businessman comes to think of his very own business — the manufacture of some particular soft drink, the production of rivets, the distilling of alcohol — as the foundation of our economic structure, so the specialist may come to believe that in his field lies every healing virtue. In my home county in southern Ohio a hundred and fifty years ago a surgeon of parts — he more than once successfully trephined skulls with a cutting instrument that he himself had made at the nearest blacksmith shop — was not so well equipped in the medical field. Once upon a time he was called to attend a little girl in an emergency while a more informed physician was being summoned. When the other doctor arrived he found the patient in convulsions, and could not guess their cause, until his predecessor explained: "Oh, that's because I gave her strychnine. I knew it would give her fits, and I'm death on fits!"

The specialist, discovering in his patient familiar symptoms, may give a secret shout of joy at finding himself on ground he knows. For the specialist like the rest of us, often flounders in ignorance. I

never submit myself to a doctor's mysterious thumpings and tappings without remembering the old railroad man, fifty years on the same job, to whom for long service a gold medal was to be presented. A special train carrying the president of the railroad and other high officials pulled up to the remote mountain siding where the old man worked. The president delivered his speech, the medal was presented, and then the old man was asked to tell something of his years of service. He had had for fifty years, he said, the same job. When a train stopped at the siding he took a long-handled hammer and went up one side of the train and down the other, tapping every wheel. "Why?" the president asked; and the old man scratched his head and said, "Well, I never did know!" That sounds ridiculous; but as a matter of fact he did not need to know; for if to one of his tappings the wheel had responded with the wrong resonance, he would have recognized the fact that something was wrong.

I sometimes suspect that any layman who approached a patient's bedside with a cheerful and reassuring manner and the conventional equipment in his bag, who went through the routine — finger on pulse and watch in hand, "Let me see your tongue, say 'Ah,' " attach a blood-pressure apparatus, listen with a stethoscope, tap here, tap there — and then said confidently: "Why you're fine. Stay in bed a couple of days and drink a lot of water" would make a highly successful practitioner — if he had sufficient common sense, when he encountered a person who was really ill, to recognize that fact and summon a consultant.

I have suggested that specialists may sometimes be too ready to see in every patient a sufferer from the ailment that is his specialty. It is perhaps a defensive psychology — for doctors are psychiatric cases too — that sometimes leads specialists to the opposite extreme. I know a man with gout who went twice to one orthopedic specialist and four times to another, complaining each time of a sore and swollen foot, before gout was even suggested! I know another man who had a sore throat and went to one doctor after another, including three throat specialists, losing his tonsils here, his adenoids there and forty pounds of weight in the process, before I told him he had thyroid disease — of which he was promptly and permanently cured.

In addition to the basic handicap of being a specialist, the specialist must overcome other disabilities. His greater knowledge may lead him contemptuously to dismiss the suggestions of the patient; yet the patient is often his own best doctor. I understand that the human body commonly manifests a sound instinct for self-defense. Does not the omentum, for instance, tend to seal off an intestinal perforation? If the jugular vein is cut, do not other veins take up its burden? If an eye or a kidney or an arm is lost, does not the remaining member rise to the occasion? The brain is an organ as intelligent as the omentum, or the jugular vein, or the kidney; and the patient's brain may conceivably know better than any specialist what the patient's body needs. So, no matter how certainly the specialist knows that his patient is wrong, he will, if he is wise, give respectful consideration to the patient's ideas.

This suggests the greatest of the many handicaps under which the specialist labors; for he does not know his patient. Lacking knowledge of the individual, he cannot accurately appraise what the patient tells him. Nor can the patient's own doctor, the general practitioner, pass on his knowledge to the specialist. He may know, yet not know how he knows. There are so many intangibles in medicine. Pain is a symptom, but what is pain? How measure it? I know a man who had an abscessed tooth that broke when an attempt was made to extract it, and who then allowed the dentist to drill out the fragments without using an anesthetic. I know another who is deterred only by shame from demanding novocain for an ordinary dental prophylaxis. Obviously, pain has one meaning for one of these men, another for the other. When a man tells his doctors that something hurts, his family doctor knows — although he cannot always say — what the man means; but the specialist does not.

So the specialist has all my sympathy. I, as a patient, — or as a member of the patient's family,— have had occasion to wish I had more of his. Once upon a time my younger son, while sliding downhill on a sled, received a hard blow on his head. Our general practitioner stitched up his scalp and the swelling subsided; but a month later, touching his head in a casual caress, I discovered a depression in his youthful skull. I called our doctor. He said comfortably, "Yes, yes." It was as though he had known or expected just this development. He assured me that there was no cause for alarm; but since I was not reassured, he suggested that I see Doctor So-and-So. Doctor So-and-So made elaborate tests and said the youngster was all right; but he took x-ray films to make sure, and told me to come in three days later — they were three long days, believe me — to hear the verdict. When I appeared at his office and gave my name, his nurse said to his receptionist, "Dr. So-and-So wishes to see Mr. Williams personally." She ushered me into an examination room with shattered skulls in the cabinets around the wall, with no chair and with nothing to read, and shut the door and left me there for forty minutes. Then Dr. So-and-So came in and said everything was fine! Now, I was glad to know that everything was fine; but to keep me waiting so long, under such circumstances, for that information was evidence of an astonishing insensibility on the part of the doctor. Nor was he an exceptional case. There was an occasion when another doctor was to telephone me, at a certain hour, the verdict of the pathologist on a bit of tissue

submitted for examination. He was three hours late in doing so, and when I spoke of this he said he had forgotten! I could cite many equally thoughtless instances, but the point needs no laboring.

Yet in such cases the doctor provokes in the patient — or protracts — a storm of fear; and fear is a poison the ill effects of which need to be recognized. Your success in quieting your patient's fear of you and his fear for himself will in many cases do more for him than all the medical knowledge accumulated through the ages. But doctors too often accentuate rather than allay the fears of their patients. They do it by putting on an act! Summoned to the bedside, they hem and they haw; they take pulse and blood pressure with carefully blank faces; they tap here and tap there, sometimes measuring with their spanned fingers or even with a tape to choose the next spot to tap, sometimes making mysterious marks with blue crayon on the cringing skin. They press a cold stethoscope against warm flesh and make the patient shiver and twitch. They ask mysterious questions without explaining why they do so. I suppose doctors had asked me two or three score times how often I had to get up at night before I mustered courage to inquire why they wanted to know. And I am not an overly timid man.

When you trot out your paraphernalia and begin your tapping and your listening, do it as though it were a matter of form — and never let the patient discover in you any slightest surprise. I stood one day in front of one of these arrangements that allows a doctor to watch what happens when the patient gulps down a tasteless white mixture. After one such swallow one of the corps of observation exclaimed, "That went almighty fast!" I am still wondering what significance attached to the speed with which that beverage went wherever it went — but I have never dared ask.

Doctors seem to wish to be mysterious. They use long words for simple things. They write prescriptions in hieroglyphics over which after the doctor is gone the patient and his family puzzle helplessly. They call a consultant and mutter and mumble with him, and then go into the hall — sometimes in the patient's sight — and whisper long together. Then in tones excessively cheerful they assure the patient that he's just fine; and they depart; and the patient and his family are left to tell each other over and over that there is nothing to worry about — and to think to themselves: "There must be something they haven't told us! Did you notice how solemn they were?"

I continue to refer to the patient and his family; for a doctor actually treats not only the patient, but those around him. The obstetrician who said he had never lost a father may have amused his listeners, but he betrayed his own complete ignorance of the extent of his responsibilities.

And these responsibilities are heavy. Not enough heed is taken by medical men of the ease with which well persons may be made sick. There are mental as well as physical contagions. When measles or scarlet fever runs through a family of children, the doctors find an easy scientific explanation: when a husband during his wife's pregnancy has morning sickness, they only smile. A doctor who read this manuscript protested that my simile was a poor one: that I compared the results of a spreading bacterial infection with an emotional situation. But there are emotional as well as physical infections. If you doubt this, read *Psychology of the Crowd*. Hitler spread an emotional infection through an entire nation.

And emotional infections may produce physical ills. Many a man, hearing a list of symptoms, imagines he has them; and the imagined may become the real. I knew one student here at Harvard Medical School who was for a while convinced that he had hydrophobia. It is true he did not contract hydrophobia; but one of his lay friends read this student's textbook on stomach ulcers, developed all the symptoms and consulted a doctor, who put him on a strict dietary regime. If service in the Army had not filled his mind with other matters, he might have become a confirmed hypochondriac.

The human imagination is a powerful force, and it is easily stimulated into activity. The wise doctor, when he puts a question, will remember that the patient is eager to give expected and pleasing replies, eager to placate in every way this doctor of whom he is secretly afraid. So he will ask his questions in such a way that the patient cannot guess the desired answer. And of course, to every reply, no matter how ominous, the wise doctor comments: "Fine! That's good!" The first time I had a life-insurance examination the doctor took my blood pressure, smiled approvingly, said "Excellent" and went through the rest of his routine. Then he said, "Now I'll make the second reading." He took my blood pressure again, and grinned. I asked the joke. "It was 184 the first time," he answered. "It's 117 now." My son recently had a physical examination for promotion in the Navy. His blood pressure was high; he was told to come in again. The second time the doctor took his blood pressure, shook his head and said, "A hundred and forty-five." Five minutes later he took it again; it was 180. He laughed and said: "Well, we'll ignore blood pressure in your case. It's obviously not a reliable indication of anything. You're scared of doctors, that's all." That was a wise doctor.

Of course some patients, discovering this terror in themselves, arm themselves against it. I knew a woman who at sixty-six consulted a doctor. He found her blood pressure astronomically high and ordered her to bed under penalty of an early demise. She said "Fiddlesticks!", spent her next twenty years in active enjoyment of life — her favorite summer recreation was digging dandelions out of

her lawn — and never again called a doctor until she was eighty-seven years old. I am sure it was only coincidence that after fourteen months of solicitous medical care she died.

Your patient is always afraid of you. Before you start your scientific tests, do all you can to reassure him. Your apparatus, you know, will increase his terror; just the sight of it. If he's a man, explain it to him, show him how it works, tell him its mechanics. I know a man with sensitive teeth who in the dentist's chair sometimes holds a mirror so that he can see what the dentist is about. Without the mirror, the dentist's drill feels as violent as one of those air hammers with which burly men rip up street pavements; the patient's tongue assures him that the cavity the dentist has excavated is enormous. But the mirror tells him the truth, quiets his fears — and helps him to forget his pain.

Interest your patient in the mechanics of your work, and if he is a man he will forget his fear of you. With women patients this is not so important. Just as men are more modest than women, so are they more imaginative and more easily frightened. Women are more interested in the doctor than in his procedures. And they are not so likely as men to be afraid. Matters of life and death are commonplace to them; they face these mysteries more calmly.

In a large proportion of cases, the good that the doctor can do the patient is determined within a few minutes after their first meeting. If you are to free the patient of his fear of you, if you are to win his confidence and if you are to enlist his co-operation, these first moments are vital. Mistakes made then are hard to remedy. The most successful doctor is the one who supplements his professional competence with what salesmen call a good approach. For before a doctor can do his best, he must sell himself to the patient, must lead the patient into such a mental attitude that he will get well to please his doctor — or to disappoint him. Tell one man he is going to die; he may retort, "I'll be damned if I do" and proceed to prove you wrong. And the reverse may sometimes be true. There are persons so contrary-minded that they will die just to prove that the doctor — who assured them they were all right — did not know what he was talking about! Any doctor of long experience will remember patients who would have recovered if they had effectively wished to do so. He will remember patients who seemed determined to die — and did.

To treat your patient properly you must within the first moments of your contact with him study the individual himself. You must decide how you will treat his mind before you decide how you will treat his body. If you are called in as a consultant, before seeing the patient at all it is wise to learn as much as possible about him from his doctor: not his symptoms, not his ailments; but what sort of man he is. Is he contrary or easily led? Intelligent or stupid? Calm or excitable? Is he frankly frightened, or does he hide his fear? For you may be sure he is afraid! What is his business? Is he a successful husband, father or friend? Has he had other illnesses? How did he take them?

As a newspaper reporter sent to interview celebrities I used to try to learn in advance something about them — and about their particular field of work — that they themselves did not know. If you can know your patient better than he knows himself, — not only physically but psychologically, — you are so much the better equipped to help him.

After you have made his acquaintance and have soothed his fears, you will already have begun, by observation, your diagnosis. If you have made a good beginning with him, he will by this time be eager to help you. Phrase your questions in such a way that you seem to expect certain answers — and do this particularly when the answer you seem to expect is not significant. If he then gives you an opposite answer, you may accept it as true; for he gave it reluctantly, believing that you expected an opposite reply and regretting that he must disappoint you. But no matter how alarming his answers to your questions may be, you should always appear to find them reassuring; for the most dangerous drug you can administer to your patient is fear.

When you reach the stage of prescribing treatment, your first care should be to avoid reawakening the fear that you have sought to relieve. As for treatment, never forget that the patient, like the customer, is always right. This may not be literally true, but it is true in enough cases to make every wise physician cautious. I remember a man who had been operated on for cancer of the rectum and who was thirsty and to whom water was forbidden. He took advantage of the nurse's momentary absence, got out of bed, walked down the corridor to the water cooler, drank all he wanted — and thereafter made an astonishingly swift recovery and never had another sick day until twenty-seven years afterward, when a runaway truck jammed him against a store front and crushed his spine — this in spite of the fact that he had had a disease that is ordinarily considered to be incurable.

But that is beside the point. The point is that he drank a forbidden draft and got better. The point is that the patient often knows best what is good for him. I remember another man who after some diagnostic procedure — I believe this was an injection of glucose to test his reaction — was told to dress, go downtown, go to a ball game and amuse himself. He protested that he felt sick and preferred to stay in bed. His nurse, presumably under instructions, insistently kidded him into going — and he collapsed in the subway, and when a taxicab brought him back to the hospital, he was in a condition so extreme that a wheel chair was necessary

to get him to his room. So the patient may have a sound idea about what treatment he needs.

To the doctor's most persistent psychiatric problem — How much of the truth shall you tell the patient? — there is no complete answer. Yet the better you know your patient, the more wisely you can solve this problem. How much of the truth will you tell a patient with a hopeless case? To be sure, you may be wrong in your diagnosis; but in what seems to you a hopeless case, will you be frank with the patient? Will you tell his family? One doctor of wide experience recently assured me that he never tells the hopeless truth. I have known two people who were killed — their deaths hastened — by being told the truth. One, an old man who had been a granite cutter in his youth, had, through a long life, been proud of the fact that he had never contracted tuberculosis. In his middle seventies he fell ill. I was with him — he was jolly, mentally himself, physically strong enough to walk and talk — when a rural doctor whom he had consulted came into the room and said flatly, "Mr. McC., you have tuberculosis." He never spoke another rational word, and died ten days later. Another, a woman, developed a progressive and incurable ailment, but her life expectancy was ten, twenty or thirty years. Told that she would never get better, she died in three months.

On the other hand, I have known men who were told that their days were numbered — and who lived full and happy lives for many a year thereafter. I think of one man who was given — on condition that he mended his outrageously dissipated ways — a maximum of six months. He decided that virtue was not worth the price, and has continued his excesses through the sixteen years that have ensued since the day sentence was pronounced on him. To decide who shall be told the hopeless truth is a problem impossible of positive solution; but he who oftenest solves it correctly is the best doctor.

Fear is a poison, yet sometimes doctors seem deliberately to provoke fear. It is as though they were completely unaware of the impact on a patient of their smallest word. A certain doctor, while making a routine examination of a patient who had come for a simple checkup, applied the stethoscope and listened for a moment and then asked gravely, "Has anyone ever told you you had heart trouble?" The patient, naturally startled, said: "No. Have I?" The doctor, without replying, turned and left the room. That was a dozen years ago, and no doctor has yet discovered that there is anything wrong with the patient's heart; but the idiotic question left behind it a cloud of shadowy terror that only time dispelled.

In another case the doctor had found puzzling symptoms. The patient spoke of a pain in his head; the doctor laughingly recalled a case of another patient who bewildered doctors for days — and turned out to have a brain tumor! So the patient acquired a new fear.

Another, a woman already in her seventies, broke her hip. When she was, so far as she would ever be, recovered, the doctor said severely: "Now you're all right; but there is one thing you must remember. Under no circumstances must you break your hip again!" So he gave her terror to walk with her all her days.

If some of these ridiculous cases seem to you incredible, I can only assure you that they are literally true. And it is the astonishing errors of great men, not the routine follies of small ones, that I have cited to you today.

It is the hard lot of the doctor to know that in the end he is always defeated; his victories at best are temporary. Death he can never finally conquer. But death's ally is fear, and this ally the doctor can defeat. Let him help the patient to conquer fear, and he will win many a skirmish; and if he can never hope to win the last grim battle, he can at least do much to rob that ultimate defeat, for his patient and for the patient's family, of the terror that is its most grievous pain.

THE INFLUENCE OF PREGNANCY ON OTOSCLEROSIS*

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THE relation of pregnancy to otosclerosis, the indication for abortion and the effect of abortion on the progress of the disease are topics that have rarely been discussed in the English medical literature. The literature in Europe in the last decade has been stimulated and influenced by German attempts to lay down rules for their eugenics courts to regulate this particular disease. In this country, no formal statements have been made on the subject, yet the interruption of a pregnancy complicating otosclerosis is not an infrequent procedure in hospitals.

MATERIAL AND METHODS

All the cases of otosclerosis at the University of Chicago Clinics were studied in conjunction with Dr. Delbert K. Judd. Those reviewed at the Massachusetts Eye and Ear Infirmary were taken largely from the extensive records of Dr. Ruth P. Guilder, of the Winthrop Foundation. This study revealed 133 otosclerotic women who had experienced one or more pregnancies. All these cases had been examined and completely tested to make the diagnosis reasonably certain. There were 19 cases in which for various reasons it was impossible to ascertain complete information concerning the effect of the pregnancies.

TABLE 1. *Effect of Pregnancy on Otosclerotic Deafness.*

DEAFNESS	NO. OF CASES	PERCENTAGE
Made worse	73	64
Unaffected	40	35
Made better	1	1
Effect unknown	19	
Total	133	

RESULTS

The results given in Table 1 demonstrate that nearly two thirds of the otosclerotics studied were made worse by at least one of their pregnancies. The cases listed as "unaffected" were those in which the course of the disease was not appreciably altered by pregnancy. These results differ from those of Schmidt,[1] who stated that in half his cases pregnancy had an unfavorable effect on otosclerosis and that in the other half there was no effect during or after it.

In only 1 case did the hearing improve during pregnancy, and this effect was only temporary. It was the patient's first pregnancy and the hearing returned to its previous level after parturition. This phenomenon has been observed in the past. Ashcroft[2] reports 2 cases of temporary improvement of otosclerotic deafness during pregnancy, one during the first three months of pregnancy only and the other during the last month. In both cases the auditory function returned to its previous level after parturition.

Many authors have blamed the progression of the disease on certain endocrine changes. Such changes taking place in one of the following periods have been suspected: during pregnancy, immediately after it (during lactation)[3, 4] and six months or more after parturition. Table 2 gives the results in this series and demonstrates that the hearing loss commences immediately after delivery in about half the cases. Whether increased deafness six months or more after delivery should be attributed to the pregnancy or to some other inciting agent is open to question.

TABLE 2. *Time of Onset of Hearing Loss.*

TIME OF ONSET	NO. OF CASES	PERCENTAGE
During pregnancy	12	20
Immediately after pregnancy (during lactation)	30	51
Six months or more after parturition	17	29
No data	14	
Total	73	

The fact that the deafness of otosclerotic women is sometimes made worse by pregnancy does not mean that it is always the first gestation that precipitates the hearing loss. Many cases have been unaffected by previous pregnancies, as demonstrated in Table 3. Fialovzky[5] states that the damage sustained in the first pregnancy in most cases recedes, whereas that aggravated by later pregnancies usually persists. Among 51 cases in the present series in which the deafness had its onset with the first pregnancy, in only 2 did the damage recede. In all the others the hearing loss was maintained. It is important to note that the

TABLE 3. *Relation of Onset of Hearing Loss to the Number of Pregnancies.*

PREGNANCY	NO. OF CASES	PERCENTAGE
First	51	70
Second	12	16
Third	5	7
Fourth	3	4
Fifth	0	0
Sixth	1	1
Seventh	1	1
Total	73	

*From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary. Presented at the annual meeting of the New England Otolaryngological Society, Boston, February 7, 1945.

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hearing may or may not be further damaged by subsequent pregnancies.

Blohmke[5] writes that the aggravation of hearing loss usually occurs not in the first pregnancy but in a later — second or third — one. He claims that this is especially true in rapidly successive childbearing. Alexander[6] and Helot[7] are of a somewhat similar opinion. Table 3 does not corroborate this, but agrees with Stein's[8] statement that the onset of the ear affection occurs most frequently at the time of the first pregnancy, although several pregnancies may occur without damaging the hearing. There was, however, little evidence in the entire series that the rapid succession of pregnancies was any more harmful to the hearing than were the same number of widely distributed gestations. Many of the otosclerotic patients who were unaffected by pregnancy experienced more than one gestation without increased hearing loss (Table 4).

TABLE 4. *Number of Pregnancies in Which Hearing was Unaffected.*

No. of Pregnancies	No. of Cases	Percentage
One	20	50
Two	12	30
Three	2	5
Four	3	8
Seven	2	5
Eleven	1	3
Total	40	

In summary, the tables demonstrate first, that many otosclerotic patients are unfavorably affected by pregnancy. This has long been known and has been observed by many medical writers. Second, that the hearing loss is likely to commence at any time in relation to pregnancy, that is during, immediately after parturition or six months later. Third, that this type of deafness most frequently has its onset with the first pregnancy, although in some cases it begins only after several gestations. Both Tables 3 and 4 indicate the first pregnancy as the one likeliest to affect the hearing (72 per cent). The patient, however, has a 50 per cent chance of experiencing subsequent pregnancies without further progression of otosclerosis.

Indications for Abortion

The problem of abortion is an extremely difficult one, for many reasons other than conflicting religious views and conflicting political ideologies. The facts that the ability to adjust to deafness psychologically varies with the individual, that otosclerosis — unlike other indications for abortion, such as toxemia, tuberculosis and heart disease — does not endanger the life of the patient and that the effect of abortion on the hearing loss is unpredictable[9–11] make every case in which this question arises controversial. As a result, the literature offers every opinion from one extreme to the other.

There are those who assert that in no case is abortion indicated in otosclerosis. Blohmke, Kümmel, Struyken and Quix[12] are among them. They believe that abortion means destroying one life for the health of another. It is their opinion, therefore, that the mother's hearing under no circumstance outweighs the baby's life. Also, Nager[13] is of the opinion that the ill effects of pregnancy on otosclerosis have been exaggerated. Of his 547 otosclerotic female patients, 264 were unmarried. If, therefore, the influence of childbearing were so deleterious, he reasons that a larger proportion of married women would have been affected. On the other hand, Welty and Brückner[12] contend that all cases of otosclerosis should be aborted. Then there are authors who have taken a stand somewhere between these extremes and have attempted the difficult task of laying down certain conditions under which abortion is justified.[14]

Haike[15] states that to propose any strict indications is impossible, but that there are certain cases in which abortion is justified. He places first those in which there has been considerable deterioration or repeated deterioration in several pregnancies. He insists, however, that this should only be done when the exact hearing status of the patient before pregnancy is known. Second, he states that abortion is especially indicated in such cases when the loss with previous pregnancies is such that another would bring on complete deafness. Finally, he mentions as an absolute indication for abortion "a torturing amount of tinnitus with psychic depression."

Goerke[9] has written that otosclerotic deafness results in an extremely severe damage to the health and that it is a justifiable indication for abortion when it renders the patient incapable of performing her occupational duties, such as teaching. He adds, however, that all alleviation of hearing loss from abortion is probably the result of self-deception; that is, the patient is relieved of uncomfortable noises and a pregnancy neurosis, a result that can sometimes be produced by physical and mental rest alone. Similar improvement is observed in some cases after delivery, as was pointed out by Alexander.[6]

In 1939, Greifenstein[16] was commissioned by the German Government to study this problem. He was instructed to review all the former indications and the clinical evidence for abortion in an attempt to add directives to the law to cover this situation. He concluded that it was impossible to assign strict directives and that clinical consideration would explode any rigid rule. He quotes Marx as writing that "one can never tell that abortion will forestall further deterioration." Greifenstein concludes his paper with the sole statement that much more material needs to be gathered before a clear decision can be given. It is of interest that he compares the incidence of therapeutic abortion

in Germany for all indications before and after Hitler. In 1932 there were 34,690 abortions, whereas in 1937 there were only 3891.

Comment

In reviewing this series, many cases were encountered that contradict most of the above opinions. It is of importance that there were many examples of multiparous otosclerotic patients who suffered a hearing loss with early pregnancies but were completely unaffected by subsequent ones. Therefore, the effect of previous pregnancies is not an accurate index of the effect of subsequent ones. The most striking fact in the entire series was the complete lack of regularity in cases of otosclerosis complicated by pregnancy. No definite rule can be laid

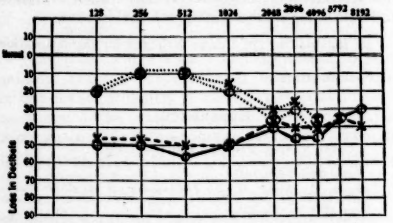

FIGURE 1. *Audiogram Taken One Year after Therapeutic Abortion and Sterilization.*

down concerning abortion until the etiology of otosclerosis is solved — if even then. Torturing tinnitus is an extremely rare by-product of otosclerotic deafness and usually represents a psychotic or neurotic problem. This can be adequately controlled by sedation and psychotherapeutic measures.

There were 2 patients in this series who had undergone a therapeutic abortion to prevent further

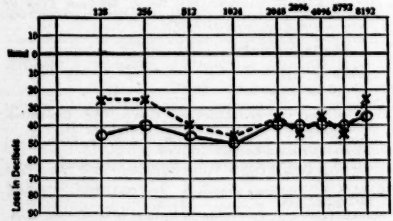

FIGURE 2. *Audiogram Taken in the Third Month of Patient's First Pregnancy, Subsequent to Therapeutic Abortion.*

progression of the deafness. One was a twenty-seven-year-old woman, whose deafness had appeared six years previously, immediately after the birth of her first child. The hearing loss was increased by a second pregnancy three years later. A year following this, the patient was aborted and sterilized by ligation of the fallopian tubes in the second month of her third pregnancy. Her hearing had not been altered by the third pregnancy and was no worse after the abortion. The patient was told that if she did not have this done, she would become totally deaf. Evidence from this series indicates this to be an unjustifiable prediction. The audiogram taken one year after the therapeutic abortion is shown in Figure 1.

The second case was that of a thirty-seven-year-old woman whose deafness came on gradually,

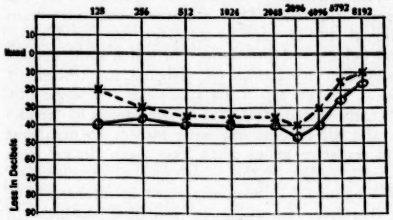

FIGURE 3. *Audiogram Taken Two Weeks after the Interruption of the Pregnancy.*

There is a slight improvement between 4096 and 8192, which was unnoticed by the patient because it is outside the speech range.

commencing twelve years previously. The maternal grandfather had had otosclerotic deafness, but no other member of the family was affected. The hearing loss progressed slowly but steadily. In November, 1940, the patient became pregnant and by the third month of pregnancy developed a marked increase in tinnitus but no apparent increase in hearing loss. An audiogram (Fig. 2) was

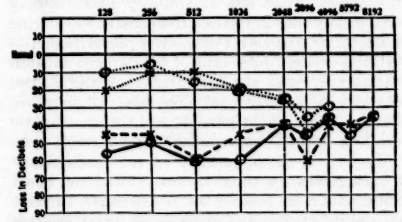

FIGURE 4. *Audiogram Taken Eight Months after the Patient's Full-Term Pregnancy and Over Three Years after the One Shown in Figure 2.*

There was a 15-decibel loss between 128 and 1024 in three years, which probably represents the natural progression of the disease. The patient noticed no hearing loss with either pregnancy.

taken at that time. An abortion, advised by an otologist to prevent further loss of function, was performed on January 17, 1941. A follow-up audiogram (Fig. 3) was taken two weeks afterward. The patient experienced satisfactory hearing with the use of a hearing aid. By the end of 1942, however, she decided to have a baby whether it damaged her hearing or not. In October, 1943, a healthy baby girl was born, and the mother noticed no change in hearing either during or after pregnancy. An audiogram (Fig. 4) taken eight months after

parturition showed a 15-decibel low-tone loss in the previous three years. The course of this patient's disease was thus apparently not unfavorably affected by a pregnancy and therapeutic abortion or by a full-term pregnancy two and a half years later.

Indications for Sterilization

The hereditary nature of otosclerosis is still being debated. The literature contains many clinical studies, such as the latest ones by Weber[17] and Schick and Goldstein,[18] in which some definite mendelian mechanism is assigned to otosclerosis. Guild,[19] however, in his studies of histologic otosclerosis, stated facts that he considered to "render invalid all conclusions, with respect to dominant and recessive genes, that have been drawn from clinical studies of the inheritance of otosclerosis." This opinion has been expressed by others.[1, 20] It is therefore impossible to prophesy deafness of progeny[9] and it would be futile to try to eliminate otosclerosis by eugenic measures,[11, 19] such as preventing propagation by otosclerotic subjects, as suggested by Guggenheim.[21]

Summary and Conclusions

Otosclerosis is a pathologic process of unknown etiology wherein new spongy bone is formed about the stapes and oval window, resulting in progressive deafness. This process is frequently unfavorably affected by pregnancy, as well as by other endocrine crises. The loss of auditory function may occur at any time in relation to pregnancy — during or after it or six months or so after parturition.

In this series, 72 per cent of the patients suffered hearing loss with the first pregnancy and 50 per cent with subsequent ones. The effect of abortion, like the effect of pregnancy, on the otosclerotic process is entirely unpredictable in the single case.

Abortion is never justified in the management of otosclerosis, for three reasons. First, the effect of pregnancy on otosclerosis is extremely variable and unpredictable, and there is no exact relation between the two conditions. The effect of previous pregnancies is not an accurate index of the effect of subsequent ones. Second, the favorable effect of abortion on the otosclerosis is also inconstant. The progression of the deafness with pregnancy may or may not be arrested by abortion. Third, the disease does not endanger the life of the mother, as do the accepted indications for therapeutic abortion, such as tuberculosis, heart disease and toxemia. Fourth, this type of deafness is not the severe handicap that it once was, owing to the advent of the modern hearing aid and the promise of surgical treatment.

Sterilization or other eugenic measures are futile in the control of otosclerosis, because the hereditary nature of the disease is not known accurately and it is impossible to prophesy deafness of progeny, and because the unfavorable effect of pregnancy on otosclerosis is not constant. In this series, the patients had a 50 per cent chance of having successive pregnancies without damage to the hearing.

The final decision concerning the role of abortion and sterilization in otosclerosis rests on a complete understanding of the etiology of the disease.

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MEDICAL PROGRESS

INDUSTRIAL HYGIENE

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ALTHOUGH the stimulus of war is no longer spurring the growth of industrial hygiene, many of the advances made during the world conflict will continue to develop. Some of the problems behind this country's industrial achievements have been brought into focus. One of these is the question of safe and healthful working conditions, and both labor and management have been fully awakened to industrial health as a factor in collective bargaining. It is to be expected that industrial medicine in the future will play a more active part in such questions. The excellent work of governmental agencies in aiding the solution of industrial-health problems is recognized by the continued growth of these organizations.[1] During the war they operated under restrictions of personnel and material, both of which are now being corrected. Undoubtedly there will be a further increase in governmental industrial-hygiene activities after peacetime reconversion is completed. At present there are forty-four official state, county and city industrial-hygiene units.

Another development in this last year of war has been the crystallization in the minds of physicians of the concept that industrial medicine is an essential part of modern practice. They are realizing the importance of industrial medicine in helping the United States to maintain its position as the most powerful industrial nation in the world. Many physicians have signified their interest in working with industry. This has been brought out by a questionnaire to Army and Navy medical personnel in which over 20 per cent of those answering indicated that they were interested in some phase of industrial medical practice.[2] To take care of the need of postgraduate courses nine universities are now prepared to supply advanced training in industrial health.[3]

Rehabilitation of the veteran continues to be one of the major post-war problems. Discharged men with physical handicaps are being competently guided back into useful life. The problem of the psychoneurotic, which is as great as that of veterans with a physical handicap, is not being overlooked.[4] The concern of American medicine with the economics of medical practice is evidenced by the number of reports on various industrial-health plans.[5–9] The features of each plan usually develop as a result of local conditions, and the further growth of medical care through the medium of industry is to be expected. During the past year tuberculosis surveys were actively promoted among industrial workers by federal and state agencies.

PNEUMOCONIOSIS

Radon has been implicated as the agent responsible for the high incidence of lung cancer in the Schneeberg and Joachimsthal mines, where it caused fifty per cent of all deaths of miners. Among contributory factors mentioned as possible etiologic agents in this mining region are dust, chronic respiratory disease, arsenic and perhaps hereditary susceptibility. A recent review of the factors by Lorenz[10] casts doubt that radium or radon has ever induced lung cancer in man. Animal experimentation and studies of other industrial workers exposed to radon do not substantiate the opinion that radioactivity produces lung cancer.

The acute toxic effects produced by quartz have been ascribed by one group of investigators to soluble silica. Others[11] have been unable to demonstrate that soluble silica is toxic, but find that the action of body fluids liberates irritating colloidal silica on the surface of quartz particles. Confirmatory evidence that colloidal silica produces toxic effects in the lungs has been furnished by perfusing lungs with this material, a specific pharmacologic bronchoconstrictor effect being thus demonstrated. Particulate silica and silica in soluble form produced no response under the same conditions. The evidence suggests that on contact with body fluids the surface of finely divided quartz may be converted to highly active colloidal silica.

Objective satisfactory tests for study of the disability arising from silicosis or other pneumoconioses have not been devised, but physiologic and clinical study provide some useful data. In a study of two groups of patients, one with silicosis and the other with siderosis, Enzer et al.[12] found that both groups showed depression of functions compared with the normal limits. This was much more marked in the latter group than in the former. The silicotic patients, however, were, on the average, thirteen years older than those with benign pneumoconiosis, which may account for the difference in physiologic response. It is significant that a depression of normal functions was found in a group of subjects suffering from so-called "benign

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pneumoconiosis." The difficulties inherent in determining disability in those suffering from pneumoconiosis is ably described by Wright.[13] Further investigative work on this problem is needed.

So-called "eggshell" calcifications have been reported in the chest films of silicotic patients, especially slate quartz miners. There is no unanimity of opinion concerning the cause of these calcified areas or even their roentgenographic distribution since some investigators maintain that they occur only in the mediastinal lymph nodes, whereas others describe them as extending into the lung parenchyma. Riemer[14] reports 4 cases occurring in workers who besides being exposed to silica dust were also exposed to limestone. He suggests that these calcifications may have been due to the inhalation of calcium.

Three acute cases of chemical pneumonitis due to exposure to beryllium were reported by Van Ordstrand et al.[15] in 1943. This metal has been identified as the causative agent in the production of a chronic pneumonitis characterized by dyspnea, orthopnea and miliary nodulation as disclosed by x-ray study and autopsy. A series of such cases occurred in Massachusetts, and confirmatory evidence is accumulating in other sections of the country where beryllium is used. Beryllium and its compounds are of increasing importance in present-day electronic manufacture and atomic research, and investigations are being conducted in an attempt to correlate acute and chronic manifestations of diseases induced by this substance.

The problem of benign pneumoconiosis has been reviewed by Pendergrass and Leopold,[16] who point out that it is impossible to differentiate the roentgen-ray appearance of silicosis or benign pneumoconiosis from that of many pulmonary diseases unassociated with the inhalation of dust. The diagnosis of such pulmonary lesions depends on a detailed knowledge of the occupational history and the environmental conditions of the worker, especially as regards the nature, concentration and particle size of the dust to which he has been exposed. The incidence of nodulation in the lung parenchyma is increasing owing to the development of manufacturing technics in which iron and other dusts are generated, for example, welding and metal grinding.

The widespread taking of x-ray photographs in industry has demonstrated numbers of people, chiefly farmers, with pulmonary miliary calcifications that apparently produce no symptoms.[17] These nodules usually consist of discrete, dense, round, uniform, shotlike spots scattered over the lungs. The causative agent has long been considered to be the tubercle bacillus, but there is some possibility that Aspergillus is the cause. *Histoplasma capsulatum* has also recently been suggested as a cause of pulmonary calcifications in the tuberculin-negative subject.[18] No data are available on the significance of these findings in relation to industrial exposure to dust.

Ritter and Nussbaum,[19, 20] in reviewing occupational illness in the cotton industry in Mississippi, state that they have been unable to find any evidence that byssinosis exists as a clinical entity. An acute febrile disease of short duration, known as "cotton fever," almost universally develops among workers when first exposed to high concentrations of cotton dust or when they return to such exposures after periods of absence. There is no evidence that chronic changes follow these manifestations. Allergic workers may develop hypersensitivity to cotton dust.

Attention has been called to a type of pneumoconiosis produced in boiler scalers, who are exposed to dust consisting of silicates, iron and salts of sodium, magnesium and calcium. The abnormal shadows seen in the x-ray films of these workers are said to be due partly to silicotic fibrosis and partly to deposits of iron dust.[21]

Aluminum Therapy and Prophylaxis in Silicosis

Powdered metallic aluminum and amorphous hydrated alumina (XH 1010) have both proved valuable in preventing silicosis in animal experimentation. Corroboration of the effectiveness of neutralizing the toxic effects of silica is evidenced by the improvement in disabled silicotic patients when treated by inhalations of either of these compounds. An organization (McIntyre Research Limited, Toronto, Canada) has been founded by the original experimenters with aluminum to develop research in silicosis control; hence, to use aluminum in this manner, a license must be obtained through this company.

There is no confirmation of the pneumoconioses described in recent years by German investigators as being caused by the inhalation of aluminum dust. Another study of pulmonary findings in workers exposed to heavy concentrations of aluminum oxide indicated no evidence of pulmonary fibrosis.[22] It must be kept in mind that the exposure of industrial workers to aluminum is not the same as that when it is used therapeutically. In industry, factors such as the specific compound, particle size and degree of concentration must be ascertained to compare toxicities.

A number of investigators have substantiated the favorable clinical results obtained by Hannon[23] on silicotic patients in the ceramic and foundry industries. In treating small groups of miners Gardner[24] did not obtain the same startling improvement, although some of his patients showed subjective improvement. Bamberger[25] in treating a group of miners obtained subjective improvement in about 50 per cent of the cases. He used both powdered metallic aluminum and hydrated alumina, and states that the latter is preferable since its

white color is less objectionable and since it appears to be more stable, does not require a bulky mill to produce it and seems to be less irritating to the respiratory tract, as evidenced by the fact that it causes less discomfort on inhalation. There is a wider experience with the use of powdered metallic aluminum, however, and it appears that the choice of either agent is a matter of personal preference.

Objective evidence of improvement in silicotic patients treated with aluminum is difficult to demonstrate, and the mechanism by which aluminum is rendered effective is not clearly understood. It is said to relieve bronchospasm, which is responsible for some of the pulmonary dysfunction. Gaseous exchange in the lungs may be interfered with by the inflammatory and immature fibrotic changes that thicken the alveolar walls of silicotic patients. Since aluminum causes regressive changes, some improvement may be due to this phenomenon. These regressive changes have not been demonstrated by x-ray.

All the data on the efficacy of aluminum as a prophylactic agent have been obtained through animal experimentation. To determine its efficacy in human beings will require many years, since silicosis does not appear on the average until ten years of exposure to a harmful concentration of the silica dust have elapsed. Engineering control should still be the recommended method for the prevention of silicosis, since dust control has the advantage of providing a cleaner work place, decreasing the psychologic hazard in the worker's mind of constant exposure to dust and diminishing the burden of the protective mechanism of the lungs in handling extraneous inhaled material. It therefore appears that at this time aluminum as a prophylactic agent should be considered only as an adjuvant to engineering control. There is no doubt that the therapeutic use of aluminum is worthy of extensive clinical application, and in several score of industrial plants aluminum is now being administered on that basis.

Industrial Poisons

So many chemicals are used in industry that it is possible to review only a fraction of those whose toxicity has been studied. It is noteworthy that lead poisoning and benzol intoxication have decreased, this being reflected in the paucity of published articles on these two substances, which were for so long pre-eminent in industrial hygiene. The decrease in lead poisoning is emphasized by Hoffman,[26] who shows a steady decline in the number of cases from 1916 to 1942. This improvement is undoubtedly due to the progress made in industrial-hygiene methods regarding protective engineering devices and regular medical examinations of workers, since the use of lead in industry has increased considerably. Occasional cases of aplastic anemia due to benzol are still reported.[27]

One of the unsolved problems in preventive industrial medicine is the development of methods to determine the early deleterious effects of exposure to chemicals. An advance in the direction of determining the early effects of hepatoxic compounds has been made. A simple test for the determination of bilirubin in the urine consists of adding five drops of 0.2 per cent aqueous methylene blue to 5 cc. of urine. If bilirubin is present, a green color is produced. This test is still experimental but has already shown its worth in the diagnosis of preicteric hepatitis.[29] Myers[29] has used it in workers exposed to tetrachlorethane and has proved its usefulness in protecting employees from further exposures to these toxic fumes.

Carbon monoxide still continues to be the most frequent cause of fatal industrial poisoning. Repeated exposure to small doses apparently induces no chronic effects. The most serious sequelae are neurologic, owing to degeneration of the brain, but occur in only a small percentage of cases following extreme exposure. The most characteristic lesion is in the lenticular nucleus, especially the globus pallidus. Experimental chronic carbon monoxide poisoning in dogs has shown that cardiac changes closely resembling those due to anoxia from other causes are produced. These changes are demonstrated by electrocardiographic variations and by morphologic changes, which include degeneration, hemorrhage and necrosis in cardiac muscle fibers. It was noted that chronic carbon monoxide intoxication in dogs occurred in concentrations that have been regarded as being within the safety limit for man.[30, 31]

Ethylene chlorhydrin, a compound frequently used in chemical synthesis and as a solvent, has been responsible for several deaths.[32, 33] The chief pathological findings were in the liver, lungs and kidney. Experimental work confirms its toxicity and the fact that it may cause death by absorption through the skin.[34] It is estimated that the lethal dose of ethylene chlorhydrin on the skin may be less than 5 cc. In all processes using this material human contact must be avoided, since there are no effective protective measures.

As the results of experiments during World War I, preventive measures were instituted at the very beginning of World War II in all plants manufacturing explosive powders. In spite of this early and planned precautionary program and a great deal of experimental research,[35] exposure to trinitrotoluene still produces occasional fatalities. Eddy[36, 37] describes 3 cases of aplastic anemia in workers exposed to only 1.5 mg. of trinitrotoluene per cubic meter of air, the accepted maximum allowable limit. During the course of the disease the patients exhibited anemia and neutropenia, and the most important symptoms were weakness, sore mouth, purpura and respiratory complaints. There is apparently an individual susceptibility.

Reactions other than those due to aplastic anemia are hepatitis, dermatosis and gastric complaints.

Methyl bromide has been finding an increasing use as a refrigerant, fumigant and insecticide. Chronic exposure to concentrations of less than 35 parts per million may cause gastrointestinal symptoms, headache and diplopia, and higher concentrations may lead to coma and convulsions.[38] On the skin it produces characteristic vesicular burns. Johnstone[39] describes an outbreak of methylene bromide intoxication in 20 packers exposed while fumigating dates. The symptomatology was chiefly referable to the central nervous system, with production of visual disturbances, ataxia, numbness of the extremities, apathy, somnolence, hallucinations, melancholia, coma, convulsions and maniacal states. In all cases there were some speech difficulty, visual disturbances and ataxia. Apparently no permanent disability resulted from these exposures.

Clinical reports of carbon tetrachloride poisoning in industry continue to appear in the literature.[40–43] Effects such as polyneuritis, uremia and nephrosis, in addition to the usual effects on the liver, are ascribed to this compound. Haggard, Miller and Greenberg,[44] in an exhaustive study of the amyl alcohols and their ketones, show the differences in the metabolic fates of seven of the eight isomers of amyl alcohol. These alcohols have a basic toxicity twelve times that of ethyl alcohol. Secondary amyl alcohol is less toxic than the primary form and more toxic than the tertiary. A study that emphasized the neurologic and mental symptoms of workers exposed to carbon bisulfide and hydrogen sulfide in the manufacture of viscose rayon has revealed no significant symptoms or signs in well controlled environments.[45]

Physical Hazards

Environmental conditions of heat and humidity in certain industries may be severer than those occurring in natural climates. The question of which temperatures cause only discomfort and inefficiency at work and which actually cause damage to health is complicated by factors of individual acclimatization and by the lack of an adequate system of correlating various combinations of temperature, humidity and air motion. All investigators of these problems agree that the wet-bulb temperature is the limiting factor in man's ability to tolerate hot environments. An exhaustive study by Eichna and his colleagues[46] determined the upper limits of environmental heat and humidity tolerated by acclimatized men in good physical condition working in hot environments. Healthy, acclimatized men work easily and efficiently when the wet-bulb temperature does not exceed 91°F. Between 91 and 94°F. they work with difficulty and inefficiently and mild heat casualties may occur. At a wet-bulb temperature of 94°F. they are incapable of sustained effort and a high incidence of heat casualties is to be expected. Dry-bulb temperatures below 120°F. have no appreciable effect.

Skinner and Pierce,[47] in evaluating environmental conditions of heat and humidity in Massachusetts, used an effective temperature of 86°F. as the maximum allowable for comfort and efficiency at work. Engineering methods are available to combat the production of high temperatures in industry, and medical methods help to control deleterious effects. Ten methods of overcoming these conditions, together with an analysis of common mistakes in engineering practice for controlling heat and humidity, are reviewed in this article. A number of experiments on the effect of vitamin depletion through sweating have been performed. Mills[48] found in studying acclimatization to tropical heat that men showed a need for almost double the dietary thiamine per unit of food and a manyfold increase in choline, which under such conditions becomes a nutritional factor of great importance. Other investigators,[49, 50] who used vitamins B_1 and C as supplementary dietary factors in workers exposed to high conditions of temperature and humidity, state that no benefit is derived from the additional intake of vitamins. The use of salt, in tablets or by salting the drinking water, to replace the chloride lost in the sweat is still basic in the treatment of physiologic disturbances from heat exposure.[51] The administration of glucose is of no advantage. Workers in hot environments maintain better efficiency if the water loss is replaced hour by hour.

Audiometric studies of workers exposed to loud noises have indicated that there may be permanent impairment of hearing. There is an awakening to this danger, although for a long time evidence has been available that fatigue, irritability, anorexia and decreased deficiency at work may be ascribed to loud noises. Engineering methods to reduce the production of noise are still inadequate, and protection must be obtained by personal protective devices.[52] Special earplugs have been developed for this purpose, although the use of cotton alone is usually sufficient to reduce the noise by five to ten decibels. This simple method frequently prevents the harmful effects of excess of noise.

Radioactive Substances

Interest in the health hazards caused by uranium has been awakened by the development of the atomic bomb. The alpha particle radioactivity of this substance has been utilized to follow its course in the body.[53] From 31 to 88 per cent is eliminated in the urine in the first twenty-four hours after intravenous injection of uranium nitrate in dogs. It was found that the administration of large doses of sodium citrate facilitated the excretion of uranium.[54, 55] This therapeutic measure was successful in causing survival in dogs receiving an otherwise

lethal dose. The effect of sodium citrate in reducing the toxic action of uranium is not completely understood, but it is suggested that its alkalizing effect does not permit development of the acid reaction necessary for precipitation and necrosis of renal protein. Further developments may be expected in the field of health hazards in atomic research, since the work occasions exposure to radioactivity in beryllium and other less known elements.

It is to be expected that chronic radium poisoning will be frequent in the next few years, since many thousands of workers were exposed because of expansion of the luminous-dial-painting industry during the war. Unfortunately, preventive measures were not universally applied, but in cases in which environmental studies were coupled with radon determinations of expired air of workers, no deleterious effects are expected. Evans, Harris and Bunker[56] produced chronic radium poisoning in rats by oral administration. Hypercalcification, bone fragility and, at the end of a year, a high incidence of osteogenic sarcoma were produced. Rats were found to be more tolerant of the radium than was man. Quantitative study showed that 4 to 8 per cent of the ingested radium is retained, and that on death an average of 3 per cent remains, over 90 per cent of this being retained in the skeleton.

Dermatitis

Although industrial dermatitis is recognized to be the most frequent disease of occupational origin, exact data on its incidence and causative factors are not available. In most states, occupational disease is not reportable, and in many of the states in which these conditions must be reported serious inadequacies exist. The most comprehensive data on the number and types of dermatitis cases in industry are presented by Brinton and Schwartz,[57] who have analyzed 32,512 cases reported by seven states for the five-year period 1938–1942. It has been observed that in certain industries the principal dermatitis-producing agent is similar to the manufactured product — notably in the manufacture of explosives, plastics, rubber, food products and leather. Industries engaged in the manufacture of metals show oils and greases to be the principal etiologic agent for dermatitis. In nearly half the industrial groups exposure to no single material existed. The dermatitis-producing agents are classified under twenty-seven headings. The most frequent causes of skin disease are petroleum products and greases (18.8 per cent), alkalis (11.7 per cent), solvents (7.8 per cent), plants and woods (6.5 per cent) and metals and metal plating (6 per cent). It is to be hoped that more accurate data will be developed as additional states require the reporting of occupational diseases and that the deficiencies of present methods will be corrected.

The allergic reactions of the skin are foremost in the interest of industrial dermatologists, since much of the skin disease in industry is due to sensitizing agents. A phenomenon that has been repeatedly observed clinically is so-called "hardening" — that is, the disappearance of an allergic contact-type dermatitis in sensitized persons on repeated exposure to the sensitizing chemical while they are still at work.[58] This finding has made it possible to continue the employment of sensitized subjects in industry and has been especially noted in workers exposed to tetryl, phenolformaldehyde and other allergens. It should be noted that hardening does not occur in all persons; that in most cases it disappears if exposure to the sensitizing chemical is discontinued; and that exposure to a great concentration of the sensitizing substance may cause recurrence of the dermatitis. One investigator questions the occurrence of this phenomenon, contending that persons showing a low degree of hypersensitiveness may continue to work under conditions of minimal exposure.

The exact place of the patch test in the diagnosis and treatment of occupational dermatitis is still subject to some controversy.[59, 60] Most authorities agree that the preplacement patch test is not a diagnostic procedure suitable to industry. In all cases in which patch tests are employed the tester must be fully familiar with all factors necessary to obtain a result capable of interpretation. Dermatophytosis and its allergic manifestations are not an important factor in lost time in industry, in spite of the wide prevalence of this disease. There is no indication that the presence of an allergy to a fungus in a worker bears any relation to the acquisition of an allergic contact dermatitis.[61]

Investigations of outbreaks of occupational dermatitis in industry continue to add to the list of industrial substances known to be primary skin irritants or sensitizers. Many of these are of great importance in war industries, especially in regard to explosives.[62] On the other hand, cutting oils, fabrics, synthetic rubber and plastics, which account for so many cases, are even more widely used in a peacetime economy. The manufacture of food products has been investigated, since contact dermatitis is the most frequent cause of occupational illness in this industry.[63] The dermatitis of fishermen, which in some cases is unique, and erysipeloid and "redfeed," which are confined almost exclusively to fishermen, have been described.[64]

Miscellaneous Conditions

Advances continue to be made in methods for the detection and quantitative determination of chemical industrial poisons. The use of the polarograph has proved of special merit, since it has the advantages of speed, precision, uniformity, relative freedom from interferences and the possibility of simultaneously determining two or more substances. Levine[65] has outlined the procedures for the determination by polarographic means of atmospheric

concentrations of lead, zinc oxide, cadmium, chromic acid and manganese.

The preventive program instituted at plants engaged in making phosphorus has eliminated the appearance of the characteristic so-called "phossy jaw," which was so frequent during World War I. It is now recognized that the earliest sign of the development of a lesion due to phosphorus is osteoporosis of the maxilla or mandible, which can be determined radiologically but is no different from that of any osteomyelitis of dental origin. Dental hygiene is extremely important, and workers who have dental extractions should be kept away from phosphorus for a period of four to six weeks to allow complete closure of the socket.[66, 67]

Leptospirosis may be considered an occupational disease, since workers exposed to water infected with rat urine show an overwhelming incidence of the disease. Thirty-seven cases were reported in Hawaii in cane cutters,[68] and in a mine in Alabama 27 cases occurred in a period of seven years. Sporadic cases in food handlers, fishermen and so forth continue to be reported in the literature. The disease is apparently amenable to treatment with penicillin if large enough doses are given. Many cases are still undiagnosed until autopsy.

* * *

The first year after the war should bring many interesting changes in industrial hygiene. A number of hazards that have been kept military secrets, such as those associated with atomic research, radar and so forth, should come to the fore. Industrial development in plastics and electronics will provide medical investigators with an opportunity to see many more cases of occupational poisoning. Some of the less tangible benefits of industrial hygiene will prove themselves to be more important as labor and management arbitrate grievances arising out of health conditions in industry.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 31411

PRESENTATION OF CASE

A seventy-seven-year-old woman was admitted to the hospital in a semicomatose condition. The history was obtained from relatives.

Two days before admission, in the morning, the patient became nauseated and vomited a considerable quantity of greenish fluid. She later complained of severe intermittent pain throughout the abdomen and in the back. On the day before admission there was no nausea or vomiting. She drank about 3 quarts of water but did not urinate all day. A bowel movement was obtained only by an enema. She was seen by a physician, who found the white-cell count to be 15,500. Later that day she became semi-stuporous. On the day of admission she passed by rectum a large amount of foul-smelling gas. In the morning the temperature was 100°F., and the pulse 120; she was admitted in the afternoon.

Her blood pressure in the past had averaged 150 systolic, 90 diastolic. For many years fatty foods had resulted in gas and mild abdominal pain. Because of her abdominal complaints, she had had numerous x-ray studies at other hospitals over the past ten years. Two diverticulums of the esophagus were found, and there were numerous diverticulums in the colon, particularly in the sigmoid. A Graham test the previous winter had revealed no stones in the gall bladder. She had had occasional episodes of mild diarrhea alternating with constipation. A number of years before admission a diagnosis of pyelitis was made, but recently the patient had complained of no dysuria, pyuria or hematuria. There had been no vaginal bleeding or discharge since the menopause.

On examination the patient was in a semiconscious state, with moderate respiratory distress. The skin was cold and clammy. The pupils were pinpoint. The lips and tongue were dry, and the former blue. The heart sounds were distant. A systolic murmur was heard at the apex and base. The pulse was weak. The lungs were clear. The abdomen was moderately distended and diffusely tender. There was tenderness in both costovertebral angles. No fluid wave was elicited, and peristalsis was normal.

The temperature was 102°F., the pulse 130, and the respirations 30. The blood pressure was 90 systolic, 45 diastolic.

Examination of the blood revealed a white-cell count of 25,000. The hemoglobin (Sahli) was 9.0 gm. per 100 cc. No urine could be obtained by catheter.

The patient continued in coma. The respirations were rapid, shallow and labored. The blood pressure remained at 90 systolic, 50 diastolic. An intravenous injection of 5 per cent dextrose in water was given with great difficulty because of collapsed veins and the fact that the patient resisted bitterly when venous puncture was attempted; indeed, the needle was placed only after she had received 4 cc. of paraldehyde intramuscularly. A whole-blood transfusion of 500 cc. was given on the evening of admission. The respirations became slower, and the patient expired fifteen hours after admission.

DIFFERENTIAL DIAGNOSIS

DR. FRANCIS D. MOORE: This is the story of a seventy-seven-year-old patient who came into the hospital in shock, and who died about sixty-three hours after the beginning of her illness. That length of elapsed time is significant, because diseases that kill people in forty-eight hours or less are often events having to do with the cardiovascular system, such as cerebral accidents, aneurysms, coronary occlusions and so on. This patient did stay alive long enough to die of one of the intra-abdominal

*On leave of absence.

catastrophes that may be treated by the surgeon. That does not rule out the other causes of reasonably sudden demise, but it does make it necessary to include consideration of peritonitis and its various causative factors.

We need not labor the question whether this patient was in shock. The blood pressure was 90 systolic, 50 diastolic, having previously been 150 systolic, 90 diastolic, and it stayed that way. She was anuric, the veins were collapsed, the pulse was weak, the heart sounds were distant and she was slightly cyanotic; it is fair to say that she was in shock.

What else did she have when she came in? She had a moderately distended and diffusely tender abdomen, which entitles one to say that she had peritonitis. A peritoneal cavity filled with blood could give that picture, but if she had had a massive intraperitoneal hemorrhage I should not have expected her to live this long. So I think that she had peritonitis and shock resulting from the peritonitis and that she died therefrom. The problem is, therefore, to find out why she had peritonitis and to see if there are any clues in the past history that might help us.

A perforated gall bladder with bile peritonitis certainly can occur in this age group, but from the evidence we have it is unlikely. A Graham test, presumably done several months before, had shown no stones in the gall bladder, and we have to accept that at its face value. There were numerous large-bowel diverticulums, particularly in the sigmoid, and she occasionally had episodes of mild diarrhea alternating with constipation. So I think that it is fair to say that she had diverticulosis, with alterations in large-bowel motility and occasional mild inflammation.

Can diverticulums in the large bowel produce the symptoms with which this woman died? Diverticulums of the sigmoid may become inflamed and perforate and cause generalized peritonitis with great rapidity. Perforated diverticulums of the sigmoid may follow other paths. After prolonged inflammation they may perforate locally and form small pericolic abscesses. These abscesses may later heal, the resulting sclerosis producing a picture of sigmoidal obstruction that is difficult to differentiate from that due to carcinoma. This patient had nothing to suggest that type of diverticulitis, and if we were to blame the final illness on diverticulitis, we should have to say that she had an open perforation into the peritoneal cavity, with no previous walling off, and massive fecal peritonitis.

There are, of course, many other things that can cause a massive peritonitis that is fatal in this short period of time. Perforation of the bowel by a foreign body, including swallowed objects, has caused rapidly fatal peritonitis. Internal hernia or other lesions involving loss of blood supply to the bowel can produce fatal peritonitis, but usually not in sixty-three hours. A perforating carcinoma of the sigmoid or an ulcer of the stomach can cause fatal peritonitis, but the latter rarely occurs in a seventy-seven-year-old woman. Such a diagnosis would have to be just a guess in this patient, as there is no evidence in the record to provide a background for a lesion of this type. Furthermore, the story is not suggestive of appendicitis.

There is one other line of evidence that I ought to dispose of, namely, the costovertebral angle tenderness. The patient was anuric and had pain in the back. Back pain, of course, can be caused by any intra-abdominal inflammatory process that involves the retroperitoneal tissues. The facts that she was anuric and that she had costovertebral tenderness are disturbing because they suggest that she had urinary obstruction and that this was somehow responsible for the apparent anuria. But on the evidence that we have I do not believe that we ought to take that too seriously. She was in severe shock with the peritoneal lesion, and the loss of body fluid into the peritoneum did not leave enough for the kidneys to excrete.

So my diagnosis is a perforated diverticulum of the large bowel, presumably of the sigmoid, although the record states that they were scattered throughout the large bowel and a cecal diverticulum can at times perforate and cause peritonitis.

DR. FREIDRICH KLEMPERER: Would you expect peristalsis to be normal in this condition?

DR. MOORE: No.

DR. KLEMPERER: It is said to have been normal.

DR. MOORE: That is correct.

A PHYSICIAN: How about pancreatitis?

DR. GORDON DONALDSON: I should like to point out the fact that this patient did have normal peristalsis when I saw her at home, and also when she came into the hospital. There was some question whether she ought to be brought to the hospital, because there was evidence of early peritonitis at home, and by the time she came in here it had spread. The trip certainly threw her into shock. All I can say is that we thought she had peritonitis and that all our efforts were directed in trying to bring her out of shock. There was never any question of operation. We spent several hours trying to give intravenous injections, and we finally succeeded in giving a transfusion. That is all we could do.

CLINICAL DIAGNOSIS

Peritonitis.

DR. MOORE'S DIAGNOSIS

Perforated sigmoidal diverticulum, with peritonitis, shock and resultant anuria.

Anatomical Diagnoses

Acute hemorrhagic pancreatitis with widespread fat necrosis.

Thrombosis of splenic, inferior mesenteric and portal veins.

Gangrene of descending colon, sigmoid and rectum.

Pathological Discussion

Dr. Benjamin Castleman: The autopsy on this patient showed about 500 cc. of thin, hemorrhagic fluid in the peritoneal cavity. There was no evidence of peritonitis, but we got the clue to the diagnosis when we saw multiple foci of fat necrosis throughout the upper abdomen. There was an extensive hemorrhagic pancreatitis, with severe necrosis of the entire body and tail. The head of the pancreas was relatively free from hemorrhage. As is almost the rule, the necrotizing process had involved radicles of the splenic vein, and in this case there was thrombosis of the main splenic vein, with retrograde extension involving the inferior mesenteric vein, so that as a complication of the pancreatitis there was venous infarction of the descending colon, sigmoid and rectum. It was the first case that I had ever seen with this type of complication, although I am sure that it must occasionally occur. The thrombosis also extended down to the mouth of the portal vein but entered it only for a short distance and had not occluded it. There was no involvement of the superior mesenteric vein, so that there was no disturbance of the blood supply to the right side of the colon. The stomach and duodenum were markedly hemorrhagic. Do you know, Dr. Donaldson, what happened the evening before the attack? Had she had a heavy meal or had she taken quite a bit of alcohol?

Dr. Donaldson: I am sure that she had not.

Dr. Castleman: A heavy meal might have caused the duodenitis. The papilla of Vater was markedly edematous, and although we did not find complete obstruction at autopsy, at one time it might have been constricted enough to have caused the pancreatitis.

Dr. Moore: Did you find any other disease of the biliary tract?

Dr. Castleman: She had a so-called "strawberry gall bladder," but there were no stones and there was free flow of bile through the common duct into the papilla at the time of autopsy. This bile was forced through by pressure on the gall bladder and, as Rich* has pointed out, this is a post-mortem procedure that may not represent the state of affairs in vivo. There was nothing significant about the kidneys, and the anuria was probably due to shock.

A Physician: How valuable is the symptomatology? The history states that for years she had been troubled with gas and abdominal pain. Is there any connection between that and the pancreatitis?

Dr. Castleman: She had cholesterosis of the gall bladder. Whether that produces symptoms at times I do not know. At the time of autopsy the gall bladder was markedly distended, so that there probably was obstruction at the papilla at one time.

Dr. Moore: That can follow pancreatitis as well as cause it. It is not infrequent.

*Rich, A. R., and Duff, G. L. Experimental and pathological studies on pathogenesis of acute haemorrhagic pancreatitis. *Bull. Johns Hopkins Hosp.* 58:212-259, 1936.

CASE 31412

Presentation of Case

First admission. A sixty-five-year-old machinist was admitted to the Emergency Ward complaining of chest pain.

The patient had had a cold and cough for several weeks. Three days before admission, while changing position in bed, he was suddenly seized by pain in the anterolateral part of the right lower chest, which was relieved by lying on his right side. There were no chills or fever, and the cough remained unchanged.

For eight years before admission, whenever he walked hurriedly, he had noted a dull aching pain starting in the left wrist and radiating upward across the shoulders and down the right arm. Nitroglycerin relieved the pain in a short time. Palpitation occurred infrequently. There had been no ankle edema at any time.

Physical examination revealed a short, stocky man in some respiratory distress. The throat was slightly injected. There was dullness to percussion over the right lung base anteriorly and posteriorly, with decreased breath sounds and moist rales. The respiratory excursions of the right chest were limited. There were moist rales at the left base, but no dullness. The heart sounds were distant. The rate was regular, and no murmurs were audible. The cardiac outline was indistinct to percussion.

The temperature was 98.4°F., the pulse 80, and the respirations 24. The blood pressure was 150 systolic, 90 diastolic.

Examination of the blood showed a white-cell count of 7800. The urine was normal.

An x-ray film of the chest taken the day before admission showed the diaphragm to be smooth in outline, with normal motion. The heart was slightly prominent in the region of the left ventricle. The aorta was tortuous. There was no definite evidence of consolidation in the lungs. A throat culture revealed a few beta-hemolytic streptococci. An electrocardiogram revealed normal rhythm, with a rate of 70, a PR interval of 0.14 second, slight depression of ST_1, inverted T_1, upright T_2 and T_3, deep, late inversion of T_4 and left-axis deviation.

The patient was discharged improved on the second hospital day.

Final admission (seventeen months later). About eighteen hours before entry the patient began to perspire profusely, and twelve hours later was seized with severe, subxiphoid pain. A physician gave him a hypodermic and sent him to the hospital.

The patient was in a shock-like condition on admission. The skin was cool, moist and pale. The blood pressure was 80 systolic, 0 diastolic. The pupils were pin point in size and did not react to light. The chest showed dullness and rales in both bases. The heart sounds were distant and weak. A friction rub was heard over the apex, and in the pulmonic area only during expiration. The pulse was weak and irregular, the rate being 120 a minute. The apical impulse was 1 cm. outside the left nipple in the fifth interspace. The abdomen was distended and tympanitic. There was slight generalized tenderness without spasm. Peristalsis was normal. The tendon reflexes were markedly reduced or absent.

Examination of the blood showed a white-cell count of 13,400, with 85 per cent neutrophils. The urine was normal. A blood Hinton test was negative. An electrocardiogram showed normal rhythm, with a rate of 120, a PR interval of 0.14 second, upright QRS_1, split QRS_2, slightly depressed ST_1, inverted T_1 and upright T_2.

The patient went steadily downhill. He vomited dark-brown guaiac-positive material. The blood pressure became unobtainable, and the heart sounds were fainter. Peristaltic sounds, which had been present two hours earlier, disappeared. He expired nine hours after admission.

Differential Diagnosis

Dr. Reed Harwood: We learn from the record of the first admission the following fairly certain facts: the patient had hypertrophy of the left ventricle, sclerosis of the coronary arteries, with angina pectoris, and probably an old inferior myocardial infarction, as suggested by the electrocardiogram. The cause of the pain that brought him to the hospital is not clear to me, and I gather from the fact that he was allowed to go home in two days that it was decided that he had nothing serious the matter with him. And as happens in so many cases in the practice of medicine, the patient recovered without the luxury of a diagnosis.

Is there anything further stated about the second electrocardiogram?

Dr. Benjamin Castleman: It is a cardiette, and only two leads are reported. Apparently they ran out of paper.

Dr. Harwood: That is too bad because I should like to know more about the T waves — particularly those in Lead 4 — and to compare them with the previous tracing.

I should also like to know how much guaiac-positive material this patient vomited.

Dr. Castleman: It says, "He vomited or retched some dark-brown guaiac-positive material." Nothing further is said about it.

Dr. Harwood: We have to deal here with the causes of severe, subxiphoid pain, shock and a rapid downhill course in a patient who has been known to have coronary heart disease. Obviously the first cause we think of is acute myocardial infarction. It is not particularly unusual to have upper abdominal pain, instead of the typical substernal pain, as a manifestation of myocardial infarction. There are several aspects of the story that make me wonder if this is the correct diagnosis. In the first place, perspiring for a period of twelve hours before the onset of pain is unusual and makes me think that something else had happened before the painful catastrophe occurred. Secondly, there was no radiation of pain to the arms. Since this patient had had pain in his arms with his attacks of angina, I should have expected a similar pain with myocardial infarction. A friction rub is an ordinary occurrence in myocardial infarction on the second or third day, but the appearance of a friction rub within eighteen hours of the onset of illness is also unusual. The electrocardiogram is not helpful in this case because it is incomplete. It may have been taken too early to record changes that would have been seen at a later date.

There is another possibility that occurs to me. He may have had a myocardial infarction a week or two previous to the onset of the pain. We should have to postulate that this was a silent infarction and that there was some unusual complication that produced death. I am thinking of a rupture of the chordae tendineae or rupture of the ventricle with cardiac tamponade. Under these circumstances I should have expected that the electrocardiogram would have shown more definite changes. Pulmonary embolism is a possibility, but we have no evidence for this, and I do not believe that it is particularly likely.

A dissecting aneurysm with cardiac tamponade is also a possibility. This case reminds me somewhat of that of a patient who was presented here a few weeks ago and who for a week before death had intermittent attacks of rather dull pain low in the neck and just below the xiphoid. With each attack of pain he had profuse perspiration. About three hours before death he developed evidence of occlusion at the bifurcation of the aorta. He died suddenly of cardiac tamponade. In the present case, there is no evidence to substantiate the diagnosis of dissecting aneurysm. There was no evidence of occluded peripheral arteries, and shock, which is usually absent in aneurysm, was present. Death occurred in a matter of hours, rather than in a matter of minutes, the latter being characteristic of cardiac tamponade.

The question to be considered is whether this patient had suffered an abdominal catastrophe to

which he succumbed as a result of his already damaged heart. There is, however, only a little evidence in favor of an abdominal emergency. The location of pain is certainly abdominal. He vomited, and there was some blood in the vomitus. He had normal peristalsis on admission, and a few hours later it was absent. There was tenderness in the abdomen. One thinks of any of the acute abdominal emergencies and could make a long list of possible diagnoses, but there is no good evidence to support any of them. I have written down ruptured viscus, acute hemorrhage, acute pancreatitis and thrombosis of some abdominal vessel. But against these diagnoses is the fact that the signs in the abdomen were not particularly definite. I should not be surprised to learn that any patient may have absent peristalsis in the last hour or two of life.

The friction rub is significant. If there actually was a friction rub, it would be hard to explain this patient's death on the basis of an abdominal catastrophe. Occasionally adventitious sounds are heard in the chest that are mistakenly diagnosed as a friction rub, but I do not believe that we have the right to suggest that in this case. If he had a friction rub, the lesion must have been in the chest. Therefore, my diagnosis is myocardial infarction. I do not know whether it was an early one, with shock and failure rapidly precipitating death, or whether it was some two or three weeks old, with a fatal complication, such as a ruptured ventricle.

Clinical Diagnosis

Coronary thrombosis, with myocardial infarction.

Dr. Harwood's Diagnosis

Myocardial infarction.

Anatomical Diagnoses

Acute hemorrhagic pancreatitis.
Coronary sclerosis.
Myocardial fibrosis.

Pathological Discussion

Dr. Castleman: The clinical diagnosis was the same as that of Dr. Harwood. In fact when this patient was brought into the Emergency Ward the medical resident pointed him out and said without knowing anything about the case, "He looks like a patient with a myocardial infarct."

At autopsy the heart weighed 450 gm., with hypertrophy predominantly of the left ventricle. In the anterior wall there was diffuse myocardial fibrosis that might be called a healed infarct. The coronary arteries were sclerotic, but there was no evidence of acute thrombosis or acute myocardial infarction.

Here again, the diagnosis was an acute hemorrhagic pancreatitis. This was much severer than that in the preceding case, involving the entire pancreas. There was no evidence of gall-bladder disease. We traced the pancreatic duct from the tail into the neck, and then it was lost. We could not find any communication between the pancreatic duct and the ampulla. The main splenic artery and veins were normal. There was some extension of the hemorrhage into the peritoneal cavity, but not so much as there was in the preceding case.

Dr. Harwood: How do you explain the pericardial friction rub?

Dr. Castleman: I do not know. Several examiners heard it, and they even charted just where in the course of the beats it occurred. It was believed to be a pleuropericardial friction rub.

Dr. Oliver Cope: The heart was large. Might that have accounted for it in any way?

Dr. Castleman: Dr. Jones, what about that?

Dr. T. Duckett Jones: I do not believe so.

Dr. Moore: What was in the peritoneal cavity? Bloody fluid?

Dr. Castleman: A small amount.

Dr. Moore: Was there fat necrosis?

Dr. Castleman: Around the pancreas and over a few areas near the lesser sac. The lesser sac contained a fair amount of blood.

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FUTURE MEDICAL AND DENTAL EDUCATION

An August 21 release by the War Manpower Commission concerns the recruitment of future medical and dental students among the veterans now being discharged from the armed services; it begins with the premise that the Nation is "facing a serious shortage of doctors and dentists in the post-war period." Our editorial eye blinked a little when confronted with this statement, for we had somehow begun to believe that with doctors, as with automobiles, the most serious lack of modern times is here and now: nothing like the numerical shortage of 1945 should recur until another war again calls us forth to battle.

The postwar period, whatever its deficiencies may be, must offer an improvement in the current supply of civilian medical and dental personnel. Both the Army and the Navy have now in their employ a much larger number of physicians and dentists than their peacetime responsibilities, including the best possible care of veterans, should call for. There will inevitably be a return to civilian life of a large number of doctors, along with other personnel. The medical-school facilities of the Nation have been producing graduates at full capacity and at top speed since September, 1941. Although the release states that some medical schools will not fill any substantial part of their first-year classes this fall, such a predicament has not arisen in this region. It must also be remembered that whatever is done to influence the supply of medical schools with students during the current year will not be effective and useful until some time after 1950. An attempt might therefore be made to formulate some clear idea concerning the needs of 1950.

When one contemplates the decades to come, with their new and ever newer remedies, machines and concepts to be applied to less prevalent diseases and, alas, to never changing human nature, one pauses as before an awesome precipice. Where shall we land if we allow ourselves to think only in terms of numbers? Our primary goal for 1950 should be not more doctors but better doctors. It is proper to have quotas for this and that and to take any measures to meet them in wartime, but wartime psychology need not project itself into the future — indeed, it is a little alarming to think of a war manpower commission that seriously concerns itself with what we all hope will be a peacetime economy. Over a long period of years the demand for medical education has been such that standards could be constantly raised. There is no reason to believe that this will not continue. A year of adjustment in which all schools will not be overflowing may occur, but this cannot be anything more than temporary and corresponds to the inevitable change-over that must be undergone by industry.

It is a good thing for the returning veterans to be aware of every opportunity that may be theirs, and they should have good advice, especially those who are contemplating the study of medicine or

dentistry. It is misleading, however, to imply that the crisis in turning out doctors and dentists is any greater than that in turning out refrigerators — or that it has any more profound or direct effect on the health of the people as a whole.

PENICILLIN IN FRIEDLÄNDER-BACILLUS INFECTIONS

A PAPER recently published records a case of *Klebsiella pneumoniae* bacteremia successfully treated by penicillin.[1] This report, and even its rather positive title, would be of only minor interest if the case had been presented with the caution, the humility and the reservations that most experienced observers express in reporting a single and inconclusive case. Instead, the authors adopt a rather provocative tone — one of challenge to the wisdom and authority of the combined experience of some of the leading clinical investigators of this country and of Great Britain. They make no attempt to analyze the course and therapy of their case to determine the place that penicillin or other therapeutic measures may have had in the recovery of their patient. They do, however, take great pains to emphasize, "The penicillin was administered in direct contradiction to all published formulae regarding the susceptibility of this organism."

It certainly was not the intention of any workers to be dogmatic or final when they classified various diseases or bacteria according to their susceptibility to penicillin; even the wide variations of different strains of the same organism are well known to such writers. Regarding infections caused by gram-negative bacilli, it is now well known that the Ducrey bacillus is relatively susceptible and clinical reports of favorable effects in the therapy of chancroid have already appeared.[2–5] An occasional strain of the influenza bacillus has also proved slightly sensitive.[6] Possibly other organisms or isolated strains may also prove to be susceptible. A careful reading of the case report under discussion, however, gives one no reason to believe that the Friedländer bacillus in that case was necessarily susceptible, nor is there any indication that the penicillin was responsible for recovery.

The patient apparently had a posterior pharyngeal abscess and some evidence of increased intracranial pressure, which, in view of the bacteremia, was interpreted as probably due to cavernous sinus phlebitis. There was profuse drainage of purulent exudate from the abscess, either spontaneous or as a result of passing a nasal tube. This drainage in itself might have been enough to account for the recovery, since the prognosis in Friedländer-bacillus bacteremias is most favorable when there is a single accessible focus of infection that can be drained. Thus, there were recoveries in 4 such cases reported from one clinic without the use of specific drugs.[7] In the present case, however, 5 gm. of sodium sulfadiazine was given intravenously soon after admission. Although it is not stated specifically whether sulfadiazine therapy was continued during the penicillin administration, it is not unlikely that more of the sulfonamide was given, and recoveries in severe cases of Friedländer-bacillus infections, including some with bacteremia, treated with sulfonamides alone have been reported.[8]

Although it may be possible that penicillin played some part in the recovery of the patient reported, particularly if there was a mixed infection in which only the Friedländer bacillus was recognized, this can by no means be considered as proved from the data presented. On the basis of this report it would be most hazardous to consider treating a bacteremic case of Friedländer-bacillus infection with penicillin alone. Since penicillin, fortunately, is relatively innocuous even when given in enormous doses, no objection to its use can be offered on that ground. There is the danger, however, of neglecting other more promising and better proved measures, such as the use of sulfadiazine and, particularly, the evacuation of accessible purulent foci; in other words, these procedures, rather than "the contradiction of all published formulae," should be stressed. Although the authors may have gained the distinction of presenting "the first record of a case of *Klebsiella pneumoniae* treated with penicillin," many other cases have undoubtedly been treated by workers who have interpreted their experiences somewhat more cautiously.

It is possible that streptomycin may prove to be effective against Friedländer-bacillus infections,

but thus far no reports concerning its clinical use are available.

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Middlesex University School of Medicine, 1934. Sponsor: Harold L. Musgrave, 620 Beach Street, Revere.

McAULIFFE, EUGENE F., JR., Boston City Hospital, Boston.
Georgetown University School of Medicine, 1943.

MOUSSELET, MABEL W., 207 Newbury Street, Boston.
Boston University School of Medicine, 1940.

POLLEN, ABRAHAM, 64 Washington Avenue, Chelsea.
Tufts College Medical School, 1940.

REYERSBACH, GERTRUD C., Massachusetts General Hospital, Boston.
University of Göttingen, 1935. Sponsor: Allan M. Butler, Massachusetts General Hospital, Boston.

RIPA, ANTHONY S., ASF Regional Hospital, Fort Ord, California.
Middlesex University School of Medicine, 1936. Sponsor: Louis Cohen, 108 Meridian Street, East Boston.

SCHIER, WOODROW W., 264 Beacon Street, Boston.
Columbia University College of Physicians and Surgeons, 1940.

SQUIRE, LUCY F., 7 Exeter Street, Boston.
Woman's Medical College of Pennsylvania, 1940.

WEXLER, JACOB, 9 Hancock Street, Boston.
Middlesex University School of Medicine, 1933. Sponsor: David B. Stearns, 416 Marlboro Street, Boston.

Robert L. Goodale, *Secretary*
330 Dartmouth Street, Boston

WORCESTER DISTRICT

CONNORS, JOHN P., 292 Lincoln Street, Worcester.
Georgetown University School of Medicine, 1930.

FITZPATRICK, AUDREY J., 2 Trinity Avenue, Worcester.
Tufts College Medical School, 1942.

FUCHS, JAKOB, 51 School Street, Milford.
University of Vienna, 1923. Sponsor: Joseph Ashkins, 36 Pine Street, Milford.

KARP, LEWIS S., 460 Park Avenue, Worcester.
Middlesex University School of Medicine, 1937. Sponsor: Joseph W. O'Connor, 36 Pleasant Street, Worcester.

KOENIG, EMIL J., JR., Reservoir Street, Holden.
Tufts College Medical School, 1940.

KRAUS, PAUL S., Grafton State Hospital, North Grafton.
University of Bologna, 1937. Sponsor: Harlan L. Paine, Grafton State Hospital, North Grafton.

ROTHSCHILD, ALFRED F., 87 Richmond Avenue, Worcester.
University of Munich, 1918. Sponsor: Joseph W. O'Connor, 36 Pleasant Street, Worcester.

Leslie P. Leland, *Secretary*
57 Cedar Street, Worcester

WORCESTER NORTH DISTRICT

PARNES, JACOB, 74th General Hospital, APO 513, c/o Postmaster, New York City (formerly: Leominster).
University of Prague Medical Faculty, 1937. Sponsor: Bartholomew P. Sweeney, 5 Gardner Place, Leominster.

James G. Simmons, *Secretary*
30 Myrtle Avenue, Fitchburg

DEATHS

BUCK — William E. Buck, M.D., of Randolph, died September 15. He was in his fifty-ninth year.
Dr. Buck received his degree from Bowdoin Medical School, in 1914.
His widow survives.

DAVISON — Arthur H. Davison, M.D., of Milton, died September 26. He was in his seventy-second year.
Dr. Davison received his degree from Harvard Medical School in 1902. He was medical director of the Boston Mutual Life Insurance Company and a former member of the Milton Hospital staff.
His widow, a daughter and a son survive.

LIBBY — J. Herbert Libby, M.D., of East Weymouth, died September 25. He was in his seventy-ninth year.
Dr. Libby received his degree from Harvard Medical School in 1892. He retired in 1942. He was a fellow of the American Medical Association.

MISCELLANY

MAJOR HALSTED AWARDED LEGION OF MERIT

Major James A. Halsted, M.C., A.U.S., on leave of absence as a member of the Medical Staff of the Massachusetts General Hospital, was recently awarded the Legion of Merit "for exceptionally meritorious conduct." The citation reads in part:

> Major Halsted, by his continuous study of the problem of gastrointestinal disorders in combat soldiers, evolved new methods of treatment which decreased the average hospitalization time from twenty-five days to six, and enabled the medical corps to return 80 per cent of such cases to full combat duty, whereas formerly only 40 per cent could be returned.

(*Notices on page xvii*)